

XP-000978832

## Effect of Macromineral Composition of Diets on Blood Acid-Base Equilibrium and Urinary Acidity in Dogs<sup>1</sup>

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### EXPANDED ABSTRACT

P.D.	PP1
p.	S93-S94 = 2

### Indexing Key Words:

• symposium • dogs • urinary pH • macrominerals • acid-base equilibrium

The importance of mineral composition in acid-base metabolism has been studied in humans for over 30 years and more recently in animals (1, 2). In dogs, however, very little is known on this subject. Understanding the impact of an acid or alkaline load on blood chemistry and urine formation could provide us useful information necessary for the adjustment of both normal and therapeutic dog diets.

**Materials and methods.** *Experiment 1: Homemade recipe with acid load.* A homemade moist basal mixture composed of (per 100 g diet) 32 g wheat whole-meal bread, 65 g lamb heart and 3 g margarine was used to prepare four diets for dogs. In diet 0, 1.5 g  $\text{CaCO}_3$ /100 g diet was added to the basal diet; in diet 1, 2.2 g  $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$ /100 g diet was added. Diet 2 was obtained by supplementing 1.1 g  $\text{CaCl}_2 \cdot 2\text{H}_2\text{O}$  and 0.7 g  $\text{CaCO}_3$ /100 g diet; in diet 3, 1.5 g  $\text{CaCO}_3$ /100 g diet and 1.6 g  $\text{NH}_4\text{Cl}$ /100 g diet were added. Mineral analysis of diets 0-3 showed a constant level of all minerals except for chloride. This resulted in a dietary undetermined anion values (dUA) of 414, 90, 272 and 73 mmol/kg dry matter in diets 0, 1, 2 and 3, respectively. Four adult dogs of different breeds between 1.5 and 9 years old were housed individually during a food adaptation period of 7 d. During the main period of 5 d, they were placed in digestibility cages. A  $4 \times 4$  Latin square design was followed. Throughout the experiment, the dogs were fed according to an energy need of 520 kJ ME/kg metabolic weight at 1130 each day. Water intake and urination (frequency, time and pH) were recorded. Thymol was used as bacterial urinary preservative, and mineral oil was put into the urine collecting

vessels to prevent gas exchanges. Blood samples were taken during three consecutive days at 1100 and 1500 h. Venous pH, base excess, bicarbonate and partial pressure of carbon dioxide were determined as measures of acid-base balance.

*Experiment 2: Commercial dry diets with alkaline load.* An identical trial was performed using three commercial dry pet diets and diet 2 from the first experiment as a reference (Table 1).

**Results.** *Experiment 1: Acid load.* Comparison of pre- and postprandial blood samples showed a severe diet-induced metabolic acidosis in groups 1 and 3 (Table 2). This was reflected in a significant depletion of the base excess. Even the fasted blood samples were lower in dogs fed diets 1 and 3 as compared with diets 0 and 2. Partial substitution of  $\text{CaCO}_3$  by  $\text{CaCl}_2$  (diet 2) induced a moderate postprandial acidosis, which was fully corrected before the next feeding. All three blood values were correlated logarithmically with dUA ( $r > 0.80$ ). All diets resulted in a typical postprandial rise in urinary pH (Table 2), which is due to gastric secretion of HCl. Adding more  $\text{CaCl}_2$  (group 1 vs. 2) reduced maximum urinary pH from 7.42 to 6.40. Peak urinary pH in the  $\text{NH}_4\text{Cl}$  group (group 3) was reached 1 hour earlier and was only 6.92. A high degree of correlation was apparent between urinary pH and dUA ( $r = 0.98/\text{Max. pH} = 5.8539 + 0.00527 \cdot \text{dUA}$ ).

*Experiment 2: Commercial diets.* Blood values for the reference diet 2 showed similar behavior as in experiment 1 (Table 3). The three commercial diets produced a slight postprandial alkalosis. In diet B, the

<sup>1</sup> Presented as part of the Waltham International Symposium on Nutrition of Small Companion Animals, at University of California, Davis, CA 95616, on September 4-8, 1990. Guest editors for the symposium were James G. Morris, D'Ann C. Finley and Quinton R. Rogers.

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TABLE 1  
Chemical Analysis of Diets in Experiment 2

Chemical analysis	Diet 2 (Exp 1)	Commercial Diets		
		A	B	C
g/100 g dry matter				
Na	0.71	1.00	0.67	1.05
K	0.68	0.41	0.22	0.42
Ca	1.46	1.34	2.01	1.53
Mg	0.10	0.18	0.15	0.23
Cl	1.34	0.59	0.67	0.75
P	0.87	1.01	1.23	1.08
S	0.25	0.15	0.11	0.27
mmol/kg dry matter				
dUA <sup>1</sup>	272	526	522	527

<sup>1</sup> dUA, dietary undetermined anion.

base excess and bicarbonate were augmented. The animals responded to this alkaline load by hypoventilation (increased pCO<sub>2</sub>). Only diet C showed no significant changes in the blood profile. The urinary pH values of diets A and B, having equal dUA, showed a remarkable resemblance with peaks of 8.30 and 8.14 (Table 3). Diet C had a lower maximum pH of 7.85,

TABLE 2  
Acid-base measurements in the plasma and urine at different levels of acid load (Experiment 1)

	Time	Diet				Pooled SEM
		0	1	2	3	
dUA		414	90	272	73	
Plasma						
Venous pH, units	1100	7.38	7.34	7.39	7.34	0.03
	1500	7.38	7.28 <sup>b</sup>	7.36 <sup>a</sup>	7.28 <sup>a</sup>	0.03
Base excess, mmol	1100	-1.36	-4.59	-1.37	-5.24	1.77
	1500	-0.45	-8.04 <sup>b</sup>	-1.93	-7.66 <sup>b</sup>	1.99
pCO <sub>2</sub> , kPa	1100	5.36	5.24	5.26	5.08	0.44
	1500	5.54	5.24	5.69 <sup>a</sup>	5.44	0.60
Bicarbonate, mmol/L	1100	23.4	20.6	23.2	19.9	1.83
	1500	24.4	18.2	21.0	18.4	2.01
Urine						
pH, units	0800	6.91	5.62	5.65	5.86	0.60
	1200	6.56	5.75	6.24	6.68	1.13
	1400	6.71	6.23	7.42	6.92	1.05
	1600	8.14	6.40	7.41	6.65	1.22
	2200	7.75	5.75	6.20	6.05	1.29

<sup>a</sup> Significantly different from respective preprandial plasma sample at  $P < 0.05$ .

<sup>b</sup> Significantly different from respective preprandial plasma sample at  $P < 0.01$ .

TABLE 3  
Acid-base measurements in the plasma and urine at different levels of alkaline load (Experiment 2)

	Time	Diet				Pooled SEM
		2	A	B	C	
dUA		272	526	522	527	
Plasma						
Venous pH, units	1100	7.36	7.36	7.36	7.34	0.02
	1500	7.33 <sup>a</sup>	7.35	7.37	7.35	0.02
Base excess, mmol/L	1100	-1.10	-1.06	-1.19	-0.80	0.69
	1500	-3.34 <sup>b</sup>	0.45	0.72 <sup>a</sup>	-0.24	1.23
pCO <sub>2</sub> , kPa	1100	5.96	5.86	5.61	6.32	0.53
	1500	6.01	6.48 <sup>b</sup>	6.31 <sup>a</sup>	6.46	0.53
Bicarbonate, mmol/L	1100	24.2	24.4	23.5	25.2	0.91
	1500	22.3 <sup>a</sup>	26.5	26.6 <sup>b</sup>	26.0	1.47
Urine						
pH, units	0800	5.93	6.64	6.73	6.55	0.46
	1200	6.52	7.18	7.21	7.78	0.64
	1400	7.62	7.13	7.24	7.87	0.63
	1600	7.69	8.14	8.30	7.57	0.45
	2200	6.30	7.58	7.65	7.16	0.59

<sup>a,b</sup> See footnote Table 2.

which occurred almost 2 hours earlier than in the other commercial diets. The correlation coefficient between dUA and the urinary pH was 0.86.

**Discussion.** Dogs, being carnivores by nature, are capable of digesting and excreting large amounts of acid. The composition of modern commercial dog foods, however, imposes an alkaline load, which makes dogs produce urine with pH values similar to those of herbivores (pH = 8). Two of three commercial diets (experiment 2) even induced slight alkaline disturbances in blood acid-base equilibrium. We therefore evaluated the possibility of adding acidifiers (e.g., CaCl<sub>2</sub>, NH<sub>4</sub>Cl) to correct food mineral imbalances. Our first trial, however, has shown that addition of these salts to a homemade diet (and perhaps also to commercial and therapeutic diets) may hold the risk of a chronic acidemia. The dUA value should be calculated and optimal values should be established.

In our opinion, dUA values between 250 and 600 mmol/kg dry matter are the safest way to obtain a physiological electrolyte balance in dogs without the risk of chronic acidemia. Further in vivo experiments are necessary to evaluate the exact importance of each of the macrominerals of the dUA value.

## LITERATURE CITED

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